EXHIBIT 1

MMWR

Recommendations and Reports

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Interpreting and Managing Blood Lead Levels <10 µg/dL in Children and Reducing Childhood Exposures to Lead:

Recommendations of CDC's Advisory Committee on Childhood Lead Poisoning Prevention

Prepared by
Advisory Committee on Childhood Lead Poisoning Prevention*

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Abstract

Lead is a common environmental contaminant, and exposure to lead is a preventable risk that exists in all areas of the United States. Lead is associated with negative outcomes in children, including impaired cognitive, motor, behavioral, and physical abilities. In 1991, CDC defined the blood lead level (BLL) that should prompt public health actions as 10 µg/dL. Concurrently, CDC also recognized that a BLL of 10 µg/dL did not define a threshold for the harmful effects of lead. Research conducted since 1991 has strengthened the evidence that children's physical and mental development can be affected at BLLs <10 µg/dL.

This report summarizes the findings of a review of clinical interpretation and management of BLLs <10 µg/dL conducted by CDC's Advisory Committee on Childhood Lead Poisoning Prevention. This report provides information to help clinicians understand BLLs <10 µg/dL, identifies gaps in knowledge concerning lead levels in this range, and outlines strategies to reduce childhood exposures to lead. In addition, this report summarizes scientific data relevant to counseling, blood lead screening, and lead exposure risk assessment.

To aid in the interpretation of BLLs, clinicians should understand the laboratory error range for blood lead values and, if possible, select a laboratory that achieves routine performance within ±2 µg/dL. Clinicians should obtain an environmental history on all children they examine, provide families with lead prevention counseling, and follow blood lead screening recommendations established for their areas. As local and patient circumstances permit, clinicians should consider early referral to developmental programs for children at high risk for exposure to lead and consider more frequent rescreening of children with BLLs approaching 10 µg/dL, depending on the potential for exposure to lead, child age, and season of testing. In addition, clinicians should direct parents to agencies and sources of information that will help them establish a lead-safe environment for

their children. For these preventive strategies to succeed, partnerships between health-care providers, families, and local public health and housing programs should be strengthened.

Introduction

Lead is a common environmental contaminant, and exposure to lead is a preventable risk in all areas of the United States. Lead is associated with negative outcomes in children, including impaired cognitive, motor, behavioral, and physical abilities (*1--4*). In 1991, CDC defined the blood lead level (BLL) that should prompt public health actions as 10 µg/dL. Concurrently, CDC also recognized that a BLL of 10 µg/dL did not define a threshold for the harmful effects of lead (*5*). Research conducted since 1991 has strengthened the evidence that children's physical and mental development can be affected at BLLs <10 µg/dL (*1,3*).

During 2002--2004, a workgroup of CDC's Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) reviewed the scientific literature regarding adverse health effects associated with BLLs <10 µg/dL, including 23 published reports that analyzed 16 separate populations with Intelligence Quotient (IQ) or general cognitive index outcomes and 12 publications related to other health outcomes. In its 2005 report, the workgroup concluded that an inverse association exists between BLLs and cognitive function, with no evidence of a weaker association in populations with lower BLLs (*1*). The direct evidence for this inverse association was strongest in a study conducted in Rochester, New York, that included children born in 1994 and1995, enrolled at age 6 months, and followed for 5 years (*6*). The majority of children studied had BLLs <10 µg/dL throughout the study period. The IQ and blood lead level relationship was most accurately described by a nonlinear negative association, with a decrease in IQ of more than seven points over the first 10 µg/dL increase in lifetime average blood lead concentration. On the basis of the evidence, the workgroup concluded that a causal association between lead exposure and impaired cognitive functioning was most likely. However, the potential for residual confounding, particularly by social factors, made the strength and shape (i.e., linear or nonlinear) of this association across BLLs uncertain. In addition, the workgroup concluded that children with BLLs <10 µg/dL should not be classified as "lead poisoned." The report noted that no safe level for blood lead in children has been identified (*1*).

Two studies published subsequently have reported negative effects of BLLs <10 µg/dL on developmental outcomes (*7,8*). One study, which included participants from the Rochester cohort (*6*) and from six other past prospective studies of children with peak BLLs across a range of values, reaffirmed an inverse association between lead at low levels and IQ (*7*). In these studies, key potential confounders were accounted for, including maternal IQ, the Home Observation for Measurement of the Environment Inventory (HOMEI) score (which is a measure of the quality and quantity of stimulation and support available to a child in the home environment), maternal education, and birth weight.

Although ACCLPP has previously reviewed case management of children with BLLs ≥10 µg/dL (*2*), this is the first ACCLPP report to summarize scientific information relevant to clinical management of children with BLLs <10 µg/dL. This report also outlines recommendations from ACCLPP to reduce childhood exposure to lead. Information on assessing an environmental history and prevention strategies to decrease exposures to lead have been published previously (*2,3*) and are not included in this report.

Methods

ACCLPP provides advice and guidance to the U.S. Department of Health and Human Services and CDC regarding new scientific knowledge and technologic developments and their practical implications for preventing childhood lead poisoning, and recommends improvements, as needed. ACCLPP members are selected on the basis of their expertise in childhood lead poisoning prevention, blood lead screening, diagnosis, and medical management. ACCLPP liaisons represent federal agencies and organizations with particular interest and expertise in childhood lead poisoning prevention.

In October 2003, ACCLPP formed another workgroup comprising three pediatricians and a CDC health scientist

to review the scientific literature regarding clinical management options for BLLs <10 µg/dL and to outline recommendations for clinical care providers. On the basis of its analysis, the workgroup developed draft recommendations that were reviewed and later adopted by ACCLPP in February 2006.

Results

Historic Trends in Children's BLLs in the United States

Since 1976, BLLs in U.S. children aged 1--5 years have decreased substantially (Table 1), primarily as a result of policies that have reduced the dispersal of lead into the environment (*9--12*). However, many U.S. children continue to be exposed to lead, primarily in their homes (*13*). Overt clinical symptoms of lead intoxication are uncommon in the United States, and lead evaluation and management strategies typically are intended to reduce the negative effects of lead on central nervous system development in children who are clinically asymptomatic.

Because no safe BLL has been defined (*1*), small reductions in population-level exposures to lead will likely affect substantial numbers of children, and can be expected to reduce the number of children affected by adverse health outcomes associated with lead exposure (*14*).

Blood Lead Measurements

As with any biologic test, blood lead measurements entail inherent uncertainties as a result of imprecise analytic techniques and preanalytic variables (e.g., the specimen collection process). However, the ratio of imprecision to measurement value, particularly at BLLs <10 µg/dL, is relatively high. The degree of inherent error in blood lead analysis varies by analytic methodology used, but whichever method is used, laboratory performance depends on the procedures and skills of the laboratory team (*15,16*). Federal regulations allow laboratories that perform blood lead testing to operate with a total allowable error of ±4 µg/dL or ±10%, whichever is greater. Consequently, at BLLs ≤10 µg/dL, a laboratory might operate within an error range of 8 µg/dL and still meet federal proficiency standards. For example, an actual value of blood lead at 7 µg/dL could be reported as being any value ranging from 3 µg/dL to 11 µg/dL and still remain within the allowable error limit. A study of duplicate testing of identical blood samples (all with a mean blood lead value <10 µg/dL) at eight laboratories reported all results as <10 µg/dL and within 3 µg/dL of the overall mean for that specimen value (*17*). A study conducted in 2006 indicated that the majority of blood lead laboratories can achieve routine performance of ±2 µg/dL at concentrations of ≤10 µg/dL without difficulty (*18*).

Blood lead test reliability also depends on adhering to blood collection techniques that reduce sample contamination. Collection of capillary blood from a fingerstick into a lead-free collection device is an accepted method for obtaining a screening test (*19--23*) and contamination by lead from the skin surface can be minimized if a protocol for proper capillary specimen collection is followed (*24*).† However, because lead levels from a capillary blood sample will vary from those of a simultaneously drawn venous sample, elevated capillary results should be confirmed with blood drawn by venipuncture. Multiple studies have reported on the uncertainty introduced by collecting capillary blood rather than venipuncture at thresholds of 10 µg/dL or 15 µg/dL (*19--23*), but none has examined the sensitivity or specificity of capillary methods at thresholds <10 µg/dL.

Children's BLL Patterns

BLLs increase quickly after an acute exposure, then gradually (over weeks) reach equilibrium with body stores of lead. Lead is distributed unevenly within the human body; in children, approximately 70% is stored in the bone compartment (*25--27*). The residence time of lead in bone can be decades (*28*). Thus, an elevated BLL will decline within a few weeks to months after an acute exposure. However, for those children with chronic lead exposure and, presumably higher bone lead stores, the decline in BLL can take much longer (*29*). Although bone lead levels can provide information regarding past absorption of lead, measurements of lead in bone using X-ray fluorescence instruments are available for research purposes only.

A newborn infant's BLL closely reflects that of the mother (*30*). During 1999--2002, the geometric mean BLL for U.S. women aged 20--59 years was 1.2 µg/dL, with 0.3% having a BLL ≥10 µg/dL (*12*). Typically, as infants become more active and increase their environmental exposures, BLLs increase. Longitudinal studies of lead-exposed children have confirmed an increase in BLLs beginning in late infancy, with a peak level reached at age 18--36 months (*6,31--33*). No studies have examined blood lead patterns specifically for children with peak levels <10 µg/dL, although certain studies have included children with levels this low. A study of children born during 1994--1995 in which >50% of the children had peak BLLs <10 µg/dL reported an expected pattern in mean BLLs of 3.4 µg/dL at age 6 months, 9.7 µg/dL at age 24 months, and 5.8 µg/dL at age 61 months (*6*). A study of children born in Boston during 1979--1981 identified mean BLLs of 7.2 µg/dL at birth, and subsequent BLLs in these children remained relatively constant (6.2 µg/dL at age 6 months, 6.8 µg/dL at age 24 months, and 6.4 µg/dL at age 57 months) (*34--36*). In both studies, higher levels of lead in home environmental samples were associated directly with higher BLLs in children (*35,37*). In addition, the Boston study demonstrated an association between the occurrence of home renovation and increased BLLs (*35*). The blood lead pattern for individual children with BLLs <10 µg/dL varies depending on their environmental exposures (*29*). More research is needed to better understand age-related patterns for BLLs that remain <10 µg/dL. However, in clinical practice, even should additional research data become available, laboratory uncertainty might interfere with a clinician's ability to detect patterns for individual children.

Once a high BLL has been established in a child, the time required for the BLL to decline to <10 µg/dL can range from months to years, depending on the duration and dose of exposure. For example, for a group of children starting at a BLL of 10--14 µg/dL and receiving case management services, the mean time required for 50% to achieve a BLL <10 µg/dL was 9 months (*38*). How much time is needed for BLLs <10 µg/dL to decline in response to interventions is unknown.

Multiple studies have confirmed that blood lead measurements vary seasonally. For example, a study conducted in Boston reported that BLLs were highest in late June and lowest in March (*39*). A Milwaukee study indicated that BLLs were higher in the summer than in the winter (*40*). Some of the variability (higher blood lead in summer) might result from increased exposure to lead in dust and soil in summer months (*41*). Blood lead values for urban children are predicted to be 1--2 mg/dL higher in the summer than winter months (*42*).

Association of BLL Patterns with Developmental Outcomes

Although BLLs peak in early childhood, when young children are especially vulnerable to lead, negative effects are associated with lead exposure at any age. Multiple studies have examined the effects of lead on children's development outcomes; in these studies, the ages at which BLLs were measured varied, as did the range of ages over which BLLs were averaged (*1--4*). Statistically significant associations have been identified between average BLLs over a specific period (e.g., 0--5 years) and various adverse health outcomes (*6,43--45*); other studies have reported statistically significant associations with a single lead measurement at a specific age (e.g., prenatal, 24 months, and 6.5 years) or with a peak measurement (*6,31,46*). Concurrent BLLs (i.e., those measured close to the time of neurodevelopmental testing) might demonstrate stronger associations with neurodevelopmental abilities than other blood lead measures (*6--8,32,47*).

Lead has a continuing negative association with IQ as children reach elementary school age. For children who participated in a trial of chelation therapy, a subsequent data analysis indicated that BLLs measured concurrently with developmental testing were associated more closely with children's cognitive abilities than was a peak level at approximately age 2 years (*48*). This association was stronger when children were tested at age 7 years than at age 5 years, which underscores the continuing need to reduce lead exposures after age 5 years.

Strategies to Enhance Children's Positive Developmental Outcomes

Although lead is a risk factor for developmental and behavior problems, its presence does not indicate that these problems will necessarily occur. No characteristic developmental pattern is attributable solely to the effects of

lead, and measures of the effects of lead on children are imperfect. Thus, for an individual child, neurobehavioral test performance might indicate clinically-significant impairments related to lead exposures but might not fully capture the array of negative outcomes caused by lead (*14*). The effects of lead at levels approaching 10 µg/dL might not be recognizable to either the child's family or clinician or be identified through neurobehavioral testing. However, lead exposure might assume greater importance for children with other environmental, genetic, biologic, social, or demographic developmental risks factors. Effects of exposures to lead at lower levels might not be evident in testing of individual children but are best evaluated on a communitywide basis (*14*).

Multiple factors influence a child's development, including how the child is treated by parents or other adult caregivers. The child's family and personal psychosocial experiences are strongly associated with performance on neurodevelopment measures and account for a greater proportion of the explained variance in these measures than BLLs <10 *m*g/dL(*2,43,45,49*). A child's blood lead measurement is estimated to account for 2%--4% of variance in neurodevelopment measures (approximately 4%--8% of the explained variance) (*2,43,50*).

All children benefit from parental nurturing, regardless of their BLL. For example, a child's language skills are enhanced by the amount of language addressed to the child (more is better), combined with a predominant pattern of positive feedback (*51*). This pattern of parenting of children under age 3 years has been associated with enhanced language and cognitive skills when children were tested in the third grade (*52*). Thus, parents might help counteract the negative effects of lead by providing a nurturing and enriched environment during development. Studies to examine effects of lead have attempted to control for this psychosocial factor by including measures such as the HOMEI score (*7*). Although no studies have specifically evaluated the effects of early intervention programs on cognitive or behavioral outcomes in relationship to children's BLLs, several laboratory studies that applied a nurturing environment to very young animals during lead acquisition demonstrated the beneficial effect of the social environment on ameliorating lead-related negative developmental outcomes (*53,54*).

Early enrichment programs, although not tested specifically in relation to BLLs, have been effective in improving cognitive development and social competence of young children, particularly infants from families with low levels of social or economic resources (*55*). Research demonstrates that children whose development has been delayed or who are at high risk for delay benefit most from interventions applied at an early age (*56--58*).

Strategies to Prevent and Reduce Exposure to Lead

CDC and the American Academy of Pediatrics (AAP) recommend that preventive care for every child should include obtaining an environmental history and identifying occupational lead exposure of household members (*2,3,5*). The major sources of lead exposure among U.S. children are lead-contaminated dust, deteriorated lead-based paint, and lead-contaminated soil (*37,59*). Typically, lead contamination of water contributes less to a child's lead burden than home and soil sources (*59*); however, if additives to water (e.g., those used in disinfection processes), are changed, the contribution of lead contamination might be greater (*60*). The extent of lead paint hazards (i.e., the presence of lead in an accessible condition, such as deteriorated lead-based paint or lead-contaminated dust or soil) on interior and exterior surfaces and in soil is associated with increased BLLs in children (*59*). Children also are exposed to nonhousing lead sources (e.g., lead in foods, cosmetics, pottery, folk remedies, and toys) (*2,3,61*).

Home-Related Lead Exposure

An estimated 4.1 million homes in the United States (25% of U.S. homes with children aged <6 years) have a lead-based paint hazard (*13*). An estimated 68% of U.S. homes built before 1940 have lead hazards, as do 43% of those built during 1940--1959 and 8% of those built during 1960--1977; estimates are higher for homes in the Northeast and Midwest and for homes in which young children reside (*13*). Despite considerable attention and resources from federal, state, and local agencies and advocacy groups, publicly available funding has not been able to provide sufficient resources to eliminate all lead paint hazards from U.S. homes. Publicly funded home inspections are most often limited to homes of children with elevated BLLs; the blood lead threshold value that prompts an inspection varies by state or municipality (*62*). In addition, even when a child's elevated BLL triggers

an inspection, public funding for repairs to reduce or eliminate identified lead hazards typically is not available.

Since 1991, lead-hazard--control grant programs through the U.S. Department of Housing and Urban Development's (HUD) Office of Healthy Homes and Lead Hazard Control (OHHLHC) have provided funding for local and state agencies to reduce lead and other environmental hazards in privately owned low-income housing. In 2005, OHHLHC allocated $139 million for this purpose, administered through seven different grant types. Other federal programs provide funding to eliminate lead-based paint hazards in federally assisted housing. Typically, the focus of these programs is on housing rehabilitation and activities that remediate lead hazards after children are identified with elevated BLLs, but HUD-funded local programs also now include primary prevention interventions that control or eliminate lead before children are exposed.

CDC is working with HUD, the U.S. Environmental Protection Agency (EPA), state and local health department lead poisoning prevention grantees, and child health and environmental justice advocates to promote primary prevention strategies to reduce exposure to lead (*1,63,64*). In addition to their traditional role of providing services to children with elevated BLLs, CDC-funded state and local lead poisoning prevention programs have been charged with implementation of housing-based primary prevention strategies in their jurisdictions. This moves beyond their traditional role of providing services to children with elevated BLLs and involves developing responses to local risks and a focus on identifying and remediating housing-based lead hazards. ACCLPP recommendations for essential elements for state and local primary prevention plans have been published previously (*63*), and strategies that have been implemented at the state and local levels to address the problem also have been outlined previously (*64*). As ACCLPP noted, implementation of state and local primary prevention plans will require 1) targeting the highest risk areas, populations, and activities; 2) fostering political will for jurisdictions to provide an adequate level of funding; 3) expanding resources for housing remediation; identification and correction of lead hazards; and 4) establishing a regulatory infrastructure to create and maintain lead-safe housing and to support the use of lead-safe construction work practices (*63,65*). Links to state and local health department web sites, which include their primary prevention plans, are available at http://www.cdc.gov/nceh/lead/grants/contracts/CLPPP%20map.htm.

Certain state and local health departments initiate case management services and home inspections when BLLs reach 10 *m*g/dL. As more primary prevention strategies are implemented, the number of health departments pursuing home inspections when BLLs reach 10 *µ*g/dL will likely increase. Certain communities have developed online registries to help parents identify homes that are lead-safe or that have lead hazards (*66*).

Steps to Identify and Safely Reduce Lead-Based Paint Hazards in Homes

Lead-based paint hazards in homes are important sources of lead exposure. Preventive actions can be implemented to identify and address these hazards. Tenants can request a copy of all lead testing reports for housing sites from landlords at any time. Their landlord should have been provided with such information when they purchased the building; compliance with a tenant request for a copy of all lead testing reports is required by federal law (*67*). In addition, federal regulations require sellers and landlords 1) to disclose the possible presence of lead-based paint in any pre-1978 property and 2) to provide information on known lead-based paint and lead-based paint hazards at the time final agreements are signed on the purchase or rental of most housing built before 1978 (e.g., by providing results of any past evaluations of the property for lead) (*67*). Prospective buyers or renters have the opportunity to arrange for a lead inspection or risk assessment by a qualified professional at their own expense; buyers have up to 10 days to check for lead. Further, the law requires sellers, landlords, and renovators to provide buyers, renters, and those hiring renovators with an EPA-approved pamphlet, "Protect Your Family from Lead in Your Home" (*68*). To protect their children from lead, parents might choose not to buy or rent a property or to negotiate remediation of identified lead hazards. However, landlords or homeowners might not know whether their property has any lead-based paint or lead hazards.

Lead-based paint hazards are likely to be present in older homes; all homes built before 1978 should be presumed either to have a lead hazard present or to contain intact lead-based paint unless a licensed lead inspector has determined otherwise. Lack of a deteriorated surface decreases the likelihood of lead-contaminated dust being present but does not ensure its absence. Knowledge of general characteristics of lead-based paint and lead-based

paint hazards and their control might help parents to understand their home better (Box) (*69--73*).

Screening for lead dust hazards through dust wipe testing (i.e., standardized collection of dust by wiping surfaces and measurement of lead collected) can help identify areas of concern. Because lead is not distributed uniformly within a home, wipe testing neither ensures absence of lead hazards at locations in the home that were not tested, nor does it ensure future protection from lead dust hazards if lead-painted surfaces subsequently deteriorate or are disturbed. Potential sources of future contamination include lead-containing paint on areas disturbed by impact/friction (e.g., windows, doors, and floors) and the interior migration of lead-contaminated exterior dust and soil (*70*). However, identifying lead dust hazards in the home is a first step toward protecting children and might help parents lower lead dust levels in their homes (*74*). Proper training is recommended for those collecting dust wipes to focus tests on areas at highest risk (*63*). Parents or property owners who wish to perform dust wipe sampling may consult their local health or housing departments for advice regarding sampling procedures, interpretation of results, and further actions based on results.

For a lead-safe environment to be established in older buildings, repair of lead hazards and careful attention to maintenance is necessary. However, local ordinances typically do not require action until a child's BLL is elevated, and property owners might be unaware of lead hazards or ignore them. Primary prevention is possible only if the focus on safety in older housing is increased and lead hazards are repaired proactively before a child is exposed. In all pre-1978 properties, owners should use lead-safe work techniques when implementing routine maintenance to decrease the likelihood of lead hazards developing in a home.

Home renovation or repair is known to be a risk factor for increasing or elevated BLLs, principally through exposures to the dust residue generated during the work (*35,75--77*). All contractors who perform repair and renovation work in older housing should be trained in lead-safe work practices and comply with any state and local requirements governing work with lead paint hazards (*78*). Property owners doing work themselves should seek expert advice and training to protect themselves and their families (*79,80*). Lead-safe work practices include 1) relocating families when the work warrants, 2) minimizing the amount of dust created, 3) containing dust in the work area, 4) cleaning up completely, 5) disposing of waste safely, and 6) performing clearance testing (i.e., testing of dust for lead after site clean up) to ensure that residual lead levels do not exceed EPA standards (*81*). Families with young children should be restricted from work areas until clearance testing has been performed and the area has been judged safe.

In previous evaluation studies, lead dust clearance standards were not low enough to protect children from increased exposures to lead-contaminated dust after lead hazard remediation; as a result, after home repairs, BLLs of children with preremediation BLLs <25 µg/dL increased (*82*). In 2001, the EPA's lead dust clearance standards were lowered to 40 $\mu g/ft^2$ for floors, 250 $\mu g/ft^2$ for windowsills, and 400 $\mu g/ft^2$ for window wells (*81*). No studies have evaluated if these lower clearance levels protect children whose BLLs are <10 µg/dL adequately from ongoing lead exposure. A cross-sectional study estimated that 20% of children with a current exposure to floor dust-lead at 40 $\mu g/ft^2$ will have BLLs ≥10 µg/dL (*83*).

A study conducted in 1994--1999 in 14 U.S. cities involving 2,682 pre-1978 homes demonstrated reductions in lead dust levels and fall in children's BLLs when lead-safe work practices were used during remediation efforts (*69,84,85*). The study applied lead dust clearance standards substantially less stringent than those currently in place, although clearance floor dust lead levels were generally low (geometric mean: 16 $\mu g/ft^2$) (*86*). However, among the 869 children in this study who were tested within 4 months before home lead remediation and approximately 7 weeks after remediation, 81 (9.3%) had a clinically significant increase (≥5 µg/dL) in BLLs; infants, children of less-educated mothers, and children from homes with higher numbers of preintervention exterior lead hazards were at highest risk (*87*). Dust lead levels at clearance were not associated significantly with an increase in BLLs. The study listed multiple types of exposures (e.g., other homes, parental job exposures) that might have accounted for increasing BLLs, but these were not evaluated systematically. Although lead remediation work reduced overall lead dust and BLLs, the finding that >9% of children had a rise in BLL of ≥5 µg/dL underscores the need to maintain a high level of vigilance to ensure that children are protected when homes or apartments undergo renovation and repair.

Educational Strategies

Lead exposure prevention strategies for children with BLLs <10 µg/dL typically focus on education and promotion of home cleanliness, without further identifying lead hazards or repairing them. Providing low-income parents with lead-related education via video in a pediatric office has been demonstrated to be effective in increasing knowledge and parental report of compliance with lead prevention actions in the home (*88*). No studies have evaluated office-based education with accompanying in-home strategies or used children's BLLs as the outcome measure for an office-based education strategy.

Studies of children at high risk that applied intervention strategies in the home or community have demonstrated the failure of education and nonprofessional cleaning conducted alone (i.e., in the absence of other measures to reduce lead exposure) in preventing the development of BLLs ≥10 µg/dL (*2,89--91*). Few studies have applied prospective designs that included control groups. One study indicated that a highly intensive education program starting at birth and lasting for ≥3 years (28 sessions) delivered by community members lowered the risk of BLLs ≥10 µg/dL 34%, but this result was not statistically significant (*92*). Repeated in-home lead prevention education, even when accompanied by complimentary supplies of cleaning materials, was ineffective in lowering the incidence of elevated BLLs (*93,94*). A review of four studies (*90*) involving caregiver education (*94,95*) and professional house cleaning (*96,97*) indicated that such low-cost interventions reduced the overall proportion of children with BLLs ≥15 µg/dL or ≥20 µg/dL, but the effect on mean BLLs was not statistically significant ($p>0.05$).

Intensive cleaning regimens reduce lead levels; in one study, biweekly professional cleaning resulted in a 17% decrease in mean BLLs after 1 year (*96*). However, the benefit of such intense and repeated cleaning was limited to homes without carpets (*98*). Intense cleaning can be used without subjecting children to a risk for increased lead exposure from unsafe repair methods (i.e., those not in compliance with lead-safe work practices). A single intensive cleaning alone does reduce levels of lead in dust by 32% to 93% depending on surface tested and starting lead concentration (*99*), but reaccumulation occurs within 3--6 months (*100,101*).

A study that involved children with BLLs 15--19 µg/dL compared the effects of nurse home visits (five visits during 1 year) accompanied by lead dust tests with those of usual care (one or two visits by an outreach worker during 1 year) (*74*). After 1 year, dust lead levels were significantly lower ($p<0.05$) in homes where lead dust tests had been conducted during intervention than in usual care homes. This finding suggests that dust testing might help parents better understand lead hazards and take action to lower them. However, changes in dust lead were not mirrored by changes in BLLs in this group of children with elevated BLLs.

Blood Lead Screening Strategies

CDC (*102*) and AAP (*3*) have recommended that health-care providers conduct blood lead tests on children enrolled in Medicaid and those identified as being at risk on the basis of the state or local screening plan or the risk assessment process. Federal policy requires that all children enrolled in Medicaid receive screening blood lead tests at ages 12 and 24 months and that blood lead screening be performed for children aged 36--72 months who have not been screened previously (*103*). Despite this, blood lead screening rates for Medicaid children have been low (<20%) (*104*) and in certain areas remain at approximately 20% (*105*). In 1997, CDC requested state and local health officials to use local communitywide data (e.g., BLL prevalence, housing age, and poverty status) to develop plans for blood lead screening for their jurisdictions and provide them to clinicians (*102*). These plans recommend either universal or targeted blood lead screening. State and local screening plans are available at http://www.cdc.gov/nceh/lead/grants/contacts/CLPPP%20Map.htm.

Targeted screening strategies enable clinicians to assess risks for individual children and recommend blood lead testing for a subset of children in the jurisdiction thought to be at increased risk for lead exposure. CDC recommends that risk evaluations be conducted on the basis of such factors as residence in a geographic area, membership in a group at high risk, answers to a personal-risk assessment questionnaire (which might include local factors such as cultural practices or products, such as herbal remedies, traditional cosmetics or imported

spices), or other risk factors relevant to the jurisdiction (*102*).

CDC recommends that locally developed targeted risk assessment and blood lead screening strategies be applied at ages 1 and 2 years (*102*). Children aged 36--72 months who have been identified as being at risk and who have not been screened previously also should receive a blood lead test (*102*). For clinicians in areas that lack a state or local screening plan, CDC recommends that a blood lead test be performed on all children at ages 1 and 2 years and on children aged 36--72 months who have not been screened previously (*102*).

Because lead exposures might change with a child's developmental progress (e.g., walking or reaching window sills) or as a result of external factors (e.g., family relocation or home remodeling), two routine screenings are recommended (at approximately ages 1 and 2 years). Among children in Chicago at high risk with BLLs <10 µg/dL at age 1 year, 21% had a BLL of ≥10 µg/dL when tested again at age ≥2 years (*103*). This report does not change current CDC recommendations in ages for routine blood lead testing. However, certain local health departments (e.g., those in Chicago, Illinois; New York, New York; and Philadelphia, Pennsylvania) recommend blood lead screening at younger ages or more frequently (*106--108*). For example, these departments recommend BLL testing starting at ages 6--9 months in high risk areas, blood lead testing at more frequent intervals (e.g., every 6 months) for children aged <2 years, or the provision of additional education and more rapid follow-up blood lead testing for children aged <12 months with BLLs 6--9 µg/dL.

Personal Lead Risk Assessment Questionnaires

The effectiveness of personal risk assessment questionnaires in identifying children with elevated BLLs has been documented in the scientific literature (Table 2) (*109--125*). However, no studies have evaluated the performance of these questionnaires at cut-off levels <10 µg/dL or their effectiveness in directing counseling or in identifying lead hazards in the home. When applied in consecutive samples of patients in clinical settings, the sensitivity of such questionnaires to identify children with BLLs ≥10 µg/dL varies considerably by population (*109--128*). In certain studies, the sensitivity improved if higher cut-off levels were used in the analysis (*103,115,119,120*) or if the questions used were developed specifically for the population tested (*113,116,117,119,120,122*). In general, to identify approximately 80% of children with BLLs ≥10 µg/dL, a blood test had to be performed for more than half of those children whose risk factors for lead exposure were assessed using a questionnaire. Multiple studies in populations with low (*109,110,112--114, 127,128*) or high (*123,124*) prevalence for elevated BLLs concluded that risk assessment questionnaires were not effective in their clinical settings.

Future Research Needs

Further study is needed to assess the effects of BLLs <10 µg/dL on children. Such research will entail following large and diverse populations, with careful attention to potential confounders and measurements of social factors. Additional research also is needed to evaluate the effectiveness of strategies to lower exposures to lead. This should include research on the effectiveness of strategies applied in the medical office and home and those that provide interventions through medical, public health, and environmental means.

Blood lead screening strategies should be evaluated to determine the most appropriate ages for screening and the utility of screening strategies applied at the community level. Evaluations of lead surveillance strategies should test ways to identify changing patterns of environmental risks and subpopulations exposed to established and emerging sources of lead. In addition, better ways should be identified to alert public and clinical health-care professionals of changes in exposure sources and patterns and to enhance their response to such changes by increased surveillance and blood lead monitoring of populations identified as being at increased risk for exposure. Additional studies might provide data that can be used to improve laboratory methods and performance monitoring. This will require developing criteria to evaluate individual laboratories and mechanisms to provide this information to clinicians.

Summary of Recommendations

For Clinicians

- Provide anticipatory guidance to parents of all young children regarding sources of lead and help them identify sources of lead in their child's environment. Obtain an environmental and family occupational history and educate parents about the most common sources of childhood lead exposure for their child and in their community. Encourage parents to identify lead hazards and sources in their homes and reduce their child's potential for exposure to lead, including the safe implementation of control measures before BLLs increase. Warn parents about the dangers poised by unsafe renovation methods and to be cognizant of the possibility of new and reemerging sources of lead in children's environments. Direct parents to local, state, and federal agencies and organizations for information, particularly concerning methods to identify and safely repair lead hazards (Appendix).
- Help parents to understand the uncertainty of a blood lead value and potential reasons for its fluctuation, including error introduced by the sampling methods and laboratory-, age-, and season-related exposures.
- Assess all children for developmental and behavior status and seek further evaluation and therapy to reduce developmental or behavioral problems, as necessary. Consider the potential influences of lead when conducting developmental screening. For children with multiple developmental risk factors, which might include lead exposures, consider more frequent developmental surveillance or conduct more extensive developmental evaluations.
- Discuss with parents the potential impact of lead on child development and promote strategies that foster optimum development, including encouraging parents to influence their child's development positively by providing nurturing and enriching experiences. For all children from economically and socially low-resource families living in areas where exposure to lead is likely, promote participation in early enrichment programs regardless of the child's BLL.
- Whenever possible, utilize laboratories that can achieve routine performance of ±2 µg/dL for blood lead analysis. Evaluate laboratory performance by reviewing the laboratory's quality control chart or statistical quality control summary.
- Review office procedures and policies to ensure that lead exposure risk assessment or blood lead screening is performed on all children as required by state or local health officials or as recommended by CDC. Consider the child's age, season of testing, and exposure history when deciding when to obtain follow-up blood lead tests. For a child whose BLL is approaching 10 µg/dL, more frequent blood lead screening (i.e., more than annually) might be appropriate, particularly if the child is aged <2 years old, was tested at the start of warm weather when BLLs tend to increase, or is at high risk for lead exposures.
- Perform a diagnostic blood lead test on all children suspected of having lead exposure or an elevated BLL and institute the recommended management guidelines if a child's BLL increases to ≥10 µg/dL.
- Become informed about lead exposure prevention strategies of local or state health departments and partner with public health agencies, community groups, and parents to work toward establishing lead-safe environments in homes and schools for all children and the reduction of exposure to lead from all sources. Advocate for the expansion of services that foster lead poisoning primary prevention.

For Government Agencies

- Increase efforts to resolve lead-based paint hazards safely before children are exposed.
- Expand services that promote lead poisoning primary prevention and develop systems that enable clinicians and parents to learn about such services.
- Develop and implement strategies to encourage the safe elimination of lead hazards in properties using trained workers and lead-safe work practices, in compliance with federal, state, and local regulations.
- Establish jurisdictional policies that mandate ensuring lead safety in housing and enforce these mandates.
- Develop and apply systematic approaches to prevent exposures to even small amounts of lead in food or consumer products, particularly when safer alternatives are available.
- Promote implementation of state and local primary prevention plans that target areas, populations, and activities of highest risk; foster political will; expand resources for housing remediation; identify and correct lead hazards; and establish a regulatory infrastructure to create and maintain lead-safe housing and

support the use of lead-safe construction work practices.
- Expand the availability of and promote the use of early enrichment programs for all children from economically and socially low-resource families living in areas where exposure to lead is likely.
- Promote and fund research that will further evaluate the effects of lead in blood at levels <10 µg/dL and evaluate strategies to identify and reduce exposure or the potential for exposure to lead, including strategies applied in medical offices and in homes.

Acknowledgment

Helpful suggestions were provided by Patrick J. Parsons, PhD, Lead Poisoning/Trace Elements Laboratory, Wadsworth Center, New York State Department of Health, Albany, New York.

References

1. CDC. Preventing lead poisoning in young children. Atlanta, GA: US Department of Health and Human Services, CDC; 2005.
2. CDC. Managing elevated BLLs among young children: recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention. Atlanta, GA: US Department of Health and Human Services, CDC; 2002. Available at http://www.cdc.gov/nceh/lead/CaseManagement/caseManage_main.htm.
3. American Academy of Pediatrics Committee on Environmental Health. Lead exposure in children: prevention, detection, and management. Pediatrics 2005;116:1036--46.
4. Bellinger DC. Lead. Pediatrics 2004;113:1016--22.
5. CDC. Preventing lead poisoning in young children. Atlanta, GA: US Department of Health and Human Services, CDC; 1991.
6. Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 µg per deciliter. N Engl J Med 2003;348: 1517--26.
7. Lanphear BP, Hornung R, Khoury J, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environ Health Perspect 2005;113:894--99.
8. Téllez-Rojo MM, Bellinger DC, Arroyo-Quiroz C, et al. Longitudinal associations between blood lead concentrations lower than 10 µg/dL and neurobehavioral development in environmentally exposed children in Mexico City. Pediatrics 2006;118:e323--30. Available at http://pediatrics.aappublications.org/content/vol118/issue2/index.shtml.
9. Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG. Chronological trend in blood lead levels between 1976 and 1980. N Engl J Med 1983;308:1373--7.
10. Pirkle JL, Brody DJ, Gunter EW, et al. The decline in blood lead levels in the United States: The National Health and Nutrition Examination Surveys (NHANES). JAMA 1994;272:284--91.
11. CDC. Blood lead levels---United States, 1991--1994. MMWR 1997;46:141--6.
12. CDC. Blood lead levels---United States, 1999--2002. MMWR 2005;54:513--6.
13. Jacobs DE, Clickner RP, Zhou JY, et al. The prevalence of lead-based paint hazards in US housing. Env Health Perspect 2002;110:A599--606.
14. Bellinger DC. What is an adverse effect? A possible resolution of clinical and epidemiological perspectives on neurobehavioral toxicity. Environ Research 2004;95:394--405.
15. Sargent JD, Johnson L, Roda S. Disparities in clinical laboratory performance for blood lead analysis. Arch Pediatr Adolesc Med 1996;150:609--14.
16. Roda SM, Greenland RD, Bornschein RL, Hammond PB. Anodic stripping voltammetry procedure modified for improved accuracy of blood lead analysis. Clin Chem 1988;34:563--7.
17. Johanputra NK, Jones R, Guckler G, et al. Accuracy and reproducibility of blood lead testing in commercial laboratories. Arch Pediatr Adolesc Med 1998;152:548--53.
18. Parsons PJ, Geraghty C, Verostek MF. An assessment of contemporary atomic spectroscopic techniques for the determination of lead in blood and urine matrices. Spectrochim Acta B 2001;56:1593--604.
19. Schlenker TL, Fritz CJ, Mark D, et al. Screening for pediatric lead poisoning. Comparability of simultaneously drawn capillary and venous blood samples. JAMA 1994;271:1346--8.
20. Parsons PJ, Reilly AA, Esernio-Jenssen D. Screening children exposed to lead: an assessment of the

capillary blood lead fingerstick test. Clin Chem 1997;43:302--11.
21. Sargent JD, Dalton MA. Rethinking the threshold for an abnormal capillary blood lead screening test. Arch Pediatr Adolesc Med 1996;150:1084--8.
22. Holtrop TG, Yee HY, Simpson PM, Kauffman RE. A community outreach lead screening program using capillary blood collected on filter paper. Arch Pediatr Adolesc Med 1998;152:455--8.
23. Schoenfeld DJ, Cullen MR, Rainey PM, et al. Screening for lead poisoning in an urban pediatric clinic using samples obtained by fingerstick. Pediatrics 1994;94:174--9.
24. CDC. Capillary blood sampling protocol. Atlanta, GA: US Department of Health and Human Services, CDC; 1997. Available at http://www.cdc.gov/nceh/lead/guide/1997/pdf/e2.pdf.
25. Barry PS, Mossman DB. Lead concentrations in human tissues. Br J Ind Med 1970;27:339--51.
26. Schroeder HA, Tipton IH. The human body burden of lead. Arch Environ Health 1968;17:965--78.
27. Leggett RW. An age-specific kinetic model of lead metabolism in humans. Environ Health Perspect 1993;101:598--616.
28. Hu H, Rabinowitz M, Smith D. Bone lead as a biological marker in epidemiologic studies of chronic toxicity: conceptual paradigms. Environ Health Perspect 1998;106:1--8.
29. Manton WI, Angle CR, Stanek KL, Reese YR, Kuehnemann TJ. Acquisition and retention of lead by young children. Environ Research 2000;82:60--80.
30. Schell LM, Denham M, Stark AD, et al. Maternal blood lead concentration, diet during pregnancy, and anthropometry predict neonatal blood lead in a socioeconomically disadvantaged population. Environ Health Perspect 2003;111:195--200.
31. Dietrich KN, Ris MD, Succop PA, Berger OG, Bornschein RL. Early exposure to lead and juvenile delinquency. Neurotoxicol Teratol 2001;23:511--8.
32. Dietrich K, Berger O, Succop P. Lead exposure and the motor developmental status of urban six-year-old children in the Cincinnati Prospective Study. Pediatrics 1993;91:301--7.
33. Baghurst PA, Robertson EF, McMichael AJ, Vimpani GV, Wibb NR, Roberts RR. The Port Pirie Cohort Study: Lead effects on pregnancy outcome and early childhood development. Neurotoxicology 1987;8:395--402.
34. Rabinowitz M, Leviton A, Needleman H. Variability of blood lead concentrations during infancy. Arch Environ Health 1984;39:74--7.
35. Rabinowitz M, Leviton A, Needleman H, Bellinger D, Waternaux C. Environmental correlates of infant blood lead levels in Boston. Environ Research 1985;38:96--107.
36. Bellinger D, Sloman J, Leviton A, Rabinowitz M, Needleman HL, Waternaux C. Low-level lead exposure and children's cognitive function in the preschool years. Pediatrics 1991;87:219--27.
37. Lanphear BP, Hornung R, Ho M, Howard CR, Eberly S, Knauf K. Environmental lead exposure during early childhood. J Pediatr 2002;140:40--7.
38. Roberts JR, Reigart JR, Ebeling M, Hulsey TC. Time required for blood lead levels to decline in nonchelated children. J Toxicol Clin Toxicol 2001;39:153--60.
39. US Environmental Protection Agency. Seasonal rhythms of blood-lead levels: Boston, 1979--1983. Washington, DC: US Environmental Protection Agency; 1995. Publication no. EPA 747-R-94--003.
40. US Environmental Protection Agency. Seasonal trends in blood lead levels in Milwaukee: statistical methodology. Washington, DC: US Environmental Protection Agency; 1996. Publication no. EPA 747-R-95-010.
41. Yiin LM, Rhoads GG, Lioy PJ. Seasonal influences on childhood lead exposure. Environ Health Perspect 2000;108:177--82.
42. Laidlaw MAS, Mielke HW, Filippelli GM, Johnson DL, Gonzales CR. Seasonality and children's blood lead levels: developing a predictive model using climatic variables and blood lead data from Indianapolis, Indiana, Syrasuse, New York, and New Orleans, Louisiana (USA). Environ Health Prespect 2005;113:793--800.
43. Wasserman GA, Liu X, Popovac D, et al. The Yugoslavia Prospective Lead Study: contributions of prenatal and postnatal lead exposure to early intelligence. Neurotoxicol Teratol 2000;22:811--8.
44. Pocock S, Smith M, Baghurst P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. BMJ 1994;309:1189--97.
45. Baghurst PA, McMichael AJ, Wigg NR, et al. Environmental exposure to lead and children's intelligence

at the age of seven years. The Port Pirie Cohort Study. N Engl J Med 1992;327:1279--84.
46. Bellinger D, Stiles K, Needleman H. Low-level lead exposure, intelligence, and academic achievement: a long-term follow-up study. Pediatrics 1992;90:855--91.
47. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 µg/dL in US children and adolescents. Public Health Rep 2000;115:521--9.
48. Chen A, Dietrich KN, Ware JH, Radcliffe J, Rogan WJ. IQ and blood lead from 2 to 7 years of age: Are the effects in older children the residual of high blood lead concentrations in 2-year-olds? Environ Health Perspect 2005;113:597--601.
49. Koller K, Brown R, Spurgeon A, Levy L. Recent developments in low-level lead exposure and intellectual impairment in children. Environ Health Perspect 2004;114:987--94.
50. Needleman H, Gatsonis C. Low-level lead exposure and the IQ of children. JAMA 1990;263:673--8.
51. Hart B, Risley TR. American parenting of language-learning children: persisting differences in family-child interactions observed in natural home environments. Dev Psychol 1992;28:1096--105.
52. Walker D. Prediction of school outcomes based on early language production and socioeconomic factors. Child Dev 1994;65:606--21.
53. Schneider JS, Lee MH, Anderson DW, Zuck L, Lidsky TI. Enriched environment during development is protective against lead-induced neurotoxicity. Brain Research 2001;896:48--55.
54. Guilarte TR, Toscano CD, McGlothan JL, Weaver SA. Environmental enrichment reverses cognitive and molecular deficits induced by developmental lead exposure. Ann Neurology 2003;53:50--6.
55. Ramey CT, Ramey SL. Prevention of intellectual disabilities: early interventions to improve cognitive development. Prev Med 1998;27:224--32.
56. Glascoe FP. Early detection of developmental and behavioral problems. Pediatr Rev 2000;21:272--80
57. Anderson LM, Shinn C, Fullilove MT, et al. The effectiveness of early childhood development programs: a systematic review. Am J Prev Med 2003;24(3 Suppl):32--46.
58. Campbell FA, Pungello EP, Miller-Johnson S, Burchinal M, Ramey CT. The development of cognitive and academic abilities: growth curves from an early childhood educational experiment. Dev Psychol 2001;37:231--42.
59. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels: a pooled analysis of 12 epidemiologic studies. Env Research 1998;79:51--68.
60. CDC. Blood lead levels in residents of homes with elevated lead in tap water---District of Columbia, 2004. MMWR 2004:53;268--70.
61. Manton WE, Angle CR, Krogstrand KLS. Origin of lead in the United States diet. Environ Sci Technol 2005;39:8995--9000.
62. National Center for Healthy Housing. Another link in the chain: state policies and practices for case management and environmental investigation for lead-poisoned children, update. Columbia, MD: National Center for Healthy Housing; 2001. Available at http://www.afhh.org/res/res_pubs/Link_in_Chain_Update.pdf.
63. CDC. Preventing lead exposure in young children: a housing-based approach to primary prevention of lead poisoning. Atlanta, GA: US Department of Health and Human Services, CDC; 2004. Available at http://www.cdc.gov/nceh/lead/Publications/Primary%20Prevention%20Document.pdf.
64. CDC. Building blocks for primary prevention: protecting children from lead-based paint hazards. Atlanta, GA: US Department of Health and Human Services, CDC; 2005. Available at http://www.cdc.gov/nceh/lead/Building%20Blocks%20June%202005.pdf.
65. Brown MJ, Gardner J, Sargent JD, Swartz K, Hu H, Timperi R. The effectiveness of housing policies in reducing children's lead exposure. Am J Public Health 2001;91:621--4.
66. US Department of Housing and Urban Development. LeadSafeHomes.info. Washington, DC: US Department of Housing and Urban Development; 2002. Available at http://www.leadsafehomes.info.
67. Lead: requirements for disclosure of known lead based paint and/or lead based paint hazards in housing, 42 U.S.C. Sect. 4852d (1992).
68. Lead-based paint poisoning prevention in certain residential structures: disclosure requirements for sellers and lessors, 40 C.F.R. Sect. 745.107 (2005).
69. National Center for Healthy Housing and University of Cincinnati Department of Environmental Health.

Evaluation of the HUD Lead-Based Paint Hazard Control Grant Program: final report. Cincinnati, OH: National Center for Healthy Housing and University of Cincinnati Department of Environmental Health; 2004. Available at http://www.hud.gov/offices/lead/EvaluationFinalReport.pdf.

70. Clark S, Menrath W, Chen M, Succop P, Bornschein R, Galke W, Wilson J. The influence of exterior dust and soil lead on interior dust lead levels in housing that had undergone lead-based paint hazard control. J Occup Environ Hyg 2004;1:273--82.
71. Dixon SL, Wilson JW, Clark CS, Galke WA, Succop PA, Chen M. The influence of common area lead loadings and lead hazard control on dust lead loadings in multiunit buildings. J Occup Environ Hyg 2005;2:659--66.
72. Binns HJ, Gray KA, Chen T, et al. Evaluation of landscape coverings to reduce soil lead hazards in urban residential yards: The Safer Yards Project. Environ Research 2004;96:127--38.
73. Yiin LM, Rhoads GG, Rich DQ, et al. Comparison of techniques to reduce residential lead dust on carpet and upholstery: The New Jersey Assessment of Cleaning Techniques Trial. Environ Health Perspect 2002;110:1233--7.
74. Brown MJ, McLaine P, Dixon S, Simon P. A randomized, community-based trial of home visiting to reduce blood lead levels in children. Pediatrics 2006;117;147--53.
75. CDC. Children with elevated blood lead levels attributed to home renovation and remodeling activities---New York, 1993--1994. MMWR 1997;45:1120--3.
76. Reissman DB, Matte TD, Gurnitz KL, Kaufmann RB, Leighton J. Is home renovation or repair a risk factor for exposure to lead among children residing in New York City? J Urban Health 2002;79:502--11.
77. US Environmental Protection Agency. Lead exposure associated with renovation and remodeling activities: phase III. Wisconsin Childhood Blood-Lead Study. Washington, DC: US Environmental Protection Agency; 1999. Publication no. EPA 747-R-99--002.
78. US Environmental Protection Agency. EPA model renovation training course minimizing lead-based paint hazards during renovation, remodeling, and painting. Washington, DC: US Environmental Protection Agency; 2000. Publication no. EPA 747-B-00-005/6.
79. US Department of Housing and Urban Development. Lead paint safety: a field guide for painting, home maintenance, and renovation work. Washington, DC: US Department of Housing and Urban Development; 2001. Available at http://www.hud.gov/offices/lead/training/LBPguide.pdf.
80. US Environmental Protection Agency. Lead in your home: a parent's reference guide. Washington, DC: US Environmental Protection Agency; 1998. Publication No. EPA 747-B-98-002. Available at http://www.epa.gov/lead/pubs/leadrev.pdf.
81. Federal Register 2001. Part III. Environmental Protection Agency. Lead; identification of dangerous levels of lead: final rule;66:120640.
82. Aschengrau A, Beiser A, Bellinger D, Copenhafer D, Weitzman D. Residential lead-based-paint hazard remediation and soil lead abatement: their impact among children with mildly elevated blood lead levels. Am J Public Health 1997;87:1698--702.
83. Lanphear BP, Weitzman M, Winter NL, et al. Lead-contaminated house dust and urban children's blood lead levels. Am J Public Health 1996;86:1416--21.
84. Galke W, Clark S, McLaine P, et al. National evaluation of the US Department of Housing and Urban Development Lead-Based Paint Hazard Control Grant Program: study methods. Environ Research 2005;98:315--28.
85. Galke W, Clark S, Wilson J, et al. Evaluation of the HUD Lead Hazard Control Grant Program: early overall findings. Environ Research 2001;86:149--56.
86. Dixon SL, Wilson JW, Succop PA, et al. Residential dust lead loading immediately after intervention in the HUD Lead Hazard Control Grant Program. J Occup Environ Hyg 2004;1:716--24.
87. Clark S, Grote JA, Wilson J, et al. Occurrence and determinants of increases in blood lead levels in children shortly after lead hazard control activities. Environ Research 2004;96:196--205.
88. Kersten HB, Moughan B, Moran MM, Spector ND, Smals LE, DeLago CW. A videotape to improve parental knowledge of lead poisoning. Ambul Pediatr 2004;4:344--7.
89. US Environmental Protection Agency. Basis for educational recommendations on reducing childhood lead exposure. Washington, DC: US Environmental Protection Agency; 2000. Publication no. EPA 747-R-00-001. Available at http://www.epa.gov/opptintr/lead/pubs/reduc_pb.pdf.

90. Haynes E, Lanphear BP, Tohn E, Farr N, Rhoads GG. The effect of interior lead hazard controls on children's blood lead concentrations: a systematic evaluation. Environ Health Perspect 2002;110:103--7.
91. Sandel M, Phelan K, Wright R, Hynes HP, Lanphear BP. The effects of housing interventions on child health. Pediatr Ann 2004;33:474--8.
92. Jordan DM, Yuse BL, Robinson LL, Hannan P, Deinard AS. A randomized trial of education to prevent lead burden in children at high risk for lead exposure: efficacy as measured by blood lead monitoring. Environ Health Perspect 2003;111:1947--51.
93. Lanphear BP, Howard C, Eberly S, et al. Primary prevention of childhood lead exposure: a randomized trial of dust control. Pediatrics 1999;103:772--7.
94. Lanphear BP, Eberly S, Howard CR. Long-term effect of dust control on blood lead concentrations. Pediatrics 2000;106:e48. Available at http://pediatrics.aappublications.org/content/vol106/issue4/index.shtml.
95. Lanphear BP, Winter NL, Apetz L, Eberly S, Weitzman M. A randomized trial of the effect of dust control on children's blood lead levels. Pediatrics 1996;98:35--40.
96. Rhoads GG, Ettinger AS, Weisel CP, et al. The effect of dust control on blood lead in toddlers: a randomized trial. Pediatrics 1999;103:551--5.
97. Aschengrau A, Hardy S, Mackey P, Pultinas D. The impact of low technology lead hazard reduction activities among children with mildly elevated blood lead levels. Environ Res 1998;79:41--50.
98. Liin LM, Lioy PJ, Rhoads GG. Impact of home carpets on childhood lead intervention study. Environ Research 2003;92:161--5.
99. Ettinger AS, Bornschein RL, Farfel M, et al. Assessment of cleaning to control lead dust in homes of children with moderate lead poisoning: Treatment of Lead-Exposed Children Trial. Environ Health Perspect 2002;110:A773--9.
100. Campbell C, Schwarz DF, Rich D, Dockery D. Effect of a follow-up professional home cleaning on serial dust and blood lead levels in urban children. Arch Environ Health 2003;58:771--80.
101. Tohn ER, Dixon SL, Wilson JW, Galke WA, Clark CS. An evaluation of one-time professional cleaning in homes with lead-based paint hazards. Appl Occup Environ Hyg 2003;18:138--43.
102. CDC. Screening young children for lead poisoning: guidance for state and local public health officials. Atlanta, GA: US Department of Health and Human Services, CDC; 1997.
103. CDC Advisory Committee on Childhood Lead Poisoning Prevention. Recommendations for blood lead screening of young children enrolled in Medicaid: targeting a group at high risk. MMWR 2000;49(No. RR-14).
104. US General Accounting Office. Lead poisoning: federal health care programs are not effectively reaching at-risk children. Washington, DC: US General Accounting Office; 1999. GAO/HEHS-99-18.
105. Kemper AR, Cohn LM, Fant KE, Dombkowski KJ. Blood lead testing among Medicaid-enrolled children in Michigan. Arch Pediatr Adolesc Med 2005;159:646--50.
106. Steinsapir C, Leighton J, Nagin D, Ehrlich J. Childhood lead poisoning prevention and management. New York City Health Information 2004;23:23--8.
107. Philadelphia Department of Public Health, Childhood Lead Poisoning Prevention Program: recommendations for the screening and management of young children potentially exposed to lead. Philadelphia, PA: Department of Public Health; 1997.
108. Chicago Department of Public Health. Blood lead testing guidelines for Chicago. Chicago, IL: Department of Public Health; 1999. Available at http://www.ci.chi.il.us/webportal/COCWebPortal/COC_EDITORIAL/ChicagoBLLTestingGuidelines.pdf.
109. Robin LF, Beller M, Middaugh JP. Statewide assessment of lead poisoning and exposure risk among children receiving Medicaid services in Alaska. Pediatrics 1997;99:e9. Available at http://pediatrics.org/cgi/content/full/99/4/e9.
110. CDC. Blood lead levels among children in a managed-care organization---California, October 1992--March 1993. MMWR 1995;44: 627--35.
111. Binns HJ, LeBailly SA, Poncher J, Kinsella TR, Saunders SE, Pediatric Practice Research Group. Is there lead in the suburbs? Risk assessment in Chicago suburban pediatric practices. Pediatrics 1994;93:164--71.
112. Kazal LA Jr. The failure of CDC screening questionnaire to efficiently detect elevated lead levels in a rural population of children. J Fam Practice 1997;45:515--8.

113. Muniz MA, Dundas R, Mahoney MC. Evaluation of a childhood lead questionnaire in predicting elevated blood lead levels in a rural community. J Rural Health 2003;19:15--9.
114. France EK, Gitterman BA, Melinkovich P, Wright RA. The accuracy of a lead questionnaire in predicting elevated pediatric blood lead levels. Arch Pediatr Adolesc Med 1996;150:958--63.
115. Binns HJ, LeBailly SA, Fingar AR, Saunders S. Evaluation of risk assessment questions used to target blood lead screening in Illinois. Pediatrics 1999;103:100--6.
116. Rooney BL, Hayes EB, Allen BK, Strutt PJ. Development of a screening tool for prediction of children at risk for lead exposure in a midwestern clinical setting. Pediatrics 1994;93:183--7.
117. Striph KB. Prevalence of lead poisoning in a suburban practice. J Fam Pract 1995;41:65--71.
118. Tejeda DM. Wyatt DD. Rostek BR. Solomon WB. Do questions about lead exposure predict elevated lead levels? Pediatrics 1994;93:192--4.
119. Snyder DC, Mohle-Boetani JC, Palla B, Fenstersheib M. Development of a population-specific risk assessment to predict elevated blood lead levels in Santa Clara County, California. Pediatrics 1995;96: 643--8.
120. Schaffer SJ, Kincaid MS, Endres N, Weitzman M. Lead poisoning risk determination in a rural setting. Pediatrics 1996;97:84--90.
121. Paulozzi LJ, Shapp J, Drawbaugh RE, Carney JK. Prevalence of lead poisoning among two-year-old children in Vermont. Pediatrics 1995;96:78--81.
122. Rolnick SJ. Nordin J, Cherney LM. A comparison of costs of universal versus targeted lead screening for young children. Environ Research 1999;80:84--91.
123. Dalton MA, Sargent JD, Stukel TA. Utility of a risk assessment questionnaire in identifying children with lead exposure. Arch Pediatr Adolesc Med 1996;150:197--202.
124. Casey R, Wiley C, Rutstein R, Pinto-Martin J. Prevalence of lead poisoning in an urban cohort of infants with high socioeconomic status. Clin Pediatr 1994;33:480--4.
125. Schaffer SJ, Szilagyi PG, Weitzman M. Lead poisoning risk determination in an urban population through the use of a standardized questionnaire. Pediatrics 1994;93:159--63.
126. Bronson MA, Tilden RL, Renier CM. Community-based screening for childhood lead poisoning. Identification of risk factors and susceptible populations in Duluth. Minn Med 1999;82:25--9.
127. Haan MN, Gerson M, Zishka BA. Identification of children at risk for lead poisoning: an evaluation of routine pediatric blood lead screening in an HMO-insured population. Pediatrics 1996;97:79--83.
128. Schonfeld DJ, Rainey PM, Cullen MR, Showalter DR, Cicchetti DV. Screening for lead poisoning by fingerstick in suburban pediatric practices. Arch Pediatr Adolesc Med 1995;149:447--50.

* A list of members of this committee appears on page 16 of this issue.

† A complimentary video or DVD entitled, "CDC Guidelines for Collecting and Handling Blood Lead Samples---2004," may be obtained from the National Center for Environmental Health, Division of Laboratory Sciences, Lead and Multielement Proficiency Program at e-mail ncehdls@cdc.gov.

Advisory Committee on Childhood Lead Poisoning Prevention

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Table 1

TABLE 1. Blood lead levels (BLLs) of children aged 1–5 years — National Health and Nutrition Examination Survey, United States, selected years

Year	% with BLL ≥10 µg/dL	Geometric mean BLL (µg/dL)
1976–1980	88.2	15.0
1991–1994	4.4	2.7
1999–2002	1.6	1.9

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Table 2

TABLE 2. Sensitivity and specificity of lead risk assessment questionnaires to predict blood lead levels (BLLs) of ≥10 µg/dL among patient samples — United States, 1994–2003

Location	Sample characteristics	Prevalence in study sample of % BLLs ≥10 µg/dL	Type of lead exposure risk assessment questions	At cut-off value of ≥10 µg/dL	
				Sensitivity	Specificity
Alaska*	Medicaid	0.6	Modified	0.83	0.39
California†	Medicaid	2.0	CDC	0.46	0.74
Suburban Chicago§	Private practices	2.2	CDC	0.69	0.70
			Modified	0.86	0.53
Arizona¶	Navajo Reservation	2.2	CDC	0.43	0.74
New York**	Rural	2.3	CDC	0.25	0.49
			Modified	0.50	NR††
Denver§§	Community health centers	2.9	Modified	0.60	0.36
Illinois¶¶	Low-risk ZIP codes	3.6	Modified	0.75	0.39
Wisconsin***	HMO Clinic A	5.4	CDC	0.77	0.37
			Modified	1.00	0.42
Ohio†††	Mixed sample	5.6	CDC	0.85	0.42
			Modified	0.92	0.57
San Francisco§§§	Mixed sample	5.8	CDC	0.87	0.75
California¶¶¶	Public clinics	6.1	CDC	0.30	0.90
			Modified	0.90	0.37
New York****	Rural	8.4	CDC	0.75	0.31
			Modified	0.88	0.44
Vermont††††	Birth certificate cohort	9.0	CDC	0.63	0.87
Minnesota§§§§	HMO	11.8	Modified	0.90	0.17
			Modified brief	0.77	0.48
Illinois¶¶¶	High-risk ZIP codes	12.1	Modified	0.74	0.27
Vermont††††	Medicaid	14.9	CDC	0.67	0.50
Wisconsin***	HMO Clinic B	16.8	CDC	0.64	0.32
			Modified	0.91	0.43
Massachusetts¶¶¶¶	Urban, high risk	21.8	CDC	0.70	0.32
Philadelphia area*****	Privately insured	29.1	CDC	0.40	0.60
Rochester, New York†††††	Primarily Medicaid	29§§§§	CDC	0.70	0.49

* **Source:** Roban LF, Seller M, Middaugh JP. Statewide assessment of lead poisoning and exposure risk among children receiving Medicaid services in Alaska. Pediatrics 1997;99:e9. Available at http://www.pediatrics.org/cgi/content/full/99/4/e9.

† **Source:** CDC. Blood lead levels among children in a managed-care organization—California, October 1992–March 1993. MMWR 1995;44:627–35.

§ **Source:** Binns HJ, LeBailly SA, Porcher J, Kmetits TR, Saunders SE, Pediatric Practice Research Group. Is there lead in the suburbs? Risk assessment in Chicago suburban pediatric practices. Pediatrics 1994;93:164–71.

¶ **Source:** Kazal LA Jr. The failure of CDC screening questionnaire to efficiently detect elevated lead levels in a rural population of children. J Fam Practice 1997;45:515–8.

** **Source:** Muntz MA, Dundas R, Mahoney MC. Evaluation of a childhood lead questionnaire in predicting elevated blood lead levels in a rural community. J Rural Health 2003;19:15–9.

†† Not reported.

§§ **Source:** France EK, Glitterman BA, Melinkovich P, Wright RA. The accuracy of a lead questionnaire in predicting elevated pediatric blood lead levels. Arch Pediatr Adolesc Med 1996;150:958–63.

¶¶ **Source:** Binns HJ, LeBailly SA, Fingar AR, Saunders S. Evaluation of risk assessment questions used to target blood lead screening in Illinois. Pediatrics 1999;103:100–6.

*** **Source:** Rooney BL, Hayes EB, Allen BK, Strutt PJ. Development of a screening tool for prediction of children at risk for lead exposure in a midwestern clinical setting. Pediatrics 1994;93:183–7.

††† **Source:** Stiph KB. Prevalence of lead poisoning in a suburban practice. J Fam Pract 1995;41:65–71.

§§§ **Source:** Tejeda DM, Wyatt DD, Rosiek SR, Solomon WB. Do questions about lead exposure predict elevated lead levels? Pediatrics 1994;93:102–4.

¶¶¶ **Source:** Snyder DC, Mohle-Boetani JC, Pelia B, Feldenshieb M. Development of a population-specific risk assessment to predict elevated blood lead levels in Santa Clara County, California. Pediatrics 1995;96:643–6.

**** **Source:** Schaffer SJ, Kincaid MS, Endres N, Weitzman M. Lead poisoning risk determination in a rural setting. Pediatrics 1996;97:94–99.

†††† **Source:** Paukkzi LJ, Sharp J, Dewhirst RE, Carney JK. Prevalence of lead poisoning among two-year-old children in Vermont. Pediatrics 1995;96:78–81.

§§§§ **Source:** Rolnick SJ, Norden J, Charney LM. A comparison of costs of universal versus targeted lead screening for young children. Environ Research 1999;80:84–91.

¶¶¶¶ **Source:** Dalton MA, Sargent JD, Stukel TA. Utility of a risk assessment questionnaire in identifying children with lead exposure. Arch Pediatr Adolesc Med 1996;150:197–202.

***** **Source:** Casey R, Wiley C, Rubstein R, Pinto-Martin J. Prevalence of lead poisoning in an urban cohort of infants with high socioeconomic status. Clin Pediatr 1994;33:480–4.

††††† **Source:** Schaffer SJ, Szilagyi PG, Weitzman M. Lead poisoning risk determination in an urban population through the use of a standardized questionnaire. Pediatrics 1994;93:159–63.

§§§§ Data not available to add a decimal place.

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Box

BOX. Tips to reduce lead-based paint and lead-based paint hazards

- Concentration of lead is generally highest in lead-based paint on exterior surfaces.
- Among interior surfaces, windows are most likely to have highest lead content.
- Interior surfaces can become contaminated from exterior sources or common areas.
- Lead-based paint on impact/friction surfaces (e.g., windows, doors, floors) deteriorates as paint is disturbed during use.
- Lack of a deteriorated surface does not ensure absence of lead-contaminated dust, although it lowers the risk.
- Renovation, remodeling, and repainting can significantly increase lead dust levels.
- Vacuum methods (using a traditional vacuum or a high-energy particulate air [HEPA] filtered vacuum) will not lower lead levels on soiled carpets or upholstery far enough to achieve safe levels.
- Creating smooth cleanable surfaces helps achieve lower dust lead levels.
- Treatments addressing lead-contaminated exterior dust/soil and building exterior lead hazards will contribute to lower lead dust in entryway and home interior locations.
- Safely addressing interior, exterior, and soil lead hazards in an integrated manner will be most beneficial in establishing lasting, lead-safe environments.

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